IN THE UNITED STATES PATENT AND TRADEMARK OFFICE

Applicants	:	KUTRYK et al.
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For	:	MEDICAL DEVICE WITH COATING THAT PROMOTES ENDOTHELIAL CELL ADHERENCE
Examiner	:	Urmi Chattopadhyay
Group Art Unit	:	3738

CERTIFICATE OF MAILING UNDER 37 C.F.R. § 1.8
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DECLARATION OF MICHAEL JOHN BRADLEY KUTRYK UNDER 37 C.F.R. § 1.132

Sirs:

1. I, Michael John Bradley Kutryk, a citizen of Canada, residing at 106 Millwood Road, Toronto, Canada M4S 1J7, Canada declare as follows:

2. I am employed as a physician and professor specializing in interventional cardiology at St. Michael's Hospital, a teaching affiliate of the University of Toronto Medical School, Toronto, Canada.

3. I have my own research laboratory at St. Michael's Hospital and have numerous publications in the field of cardiovascular research. My curriculum vitae is attached (Exhibit 1).

4. I am also a co-inventor of the invention disclosed by and claimed in the referenced application. As such, I am familiar with the state of the art at the time the invention was made and have direct knowledge of the referenced application and its prosecution history.

5. I understand that the Examiner has issued a final Office Action in the referenced application and has rejected the pending claims as obvious over the publication to Dekker A., et al., *Thrombosis and Haemostasis*, "Improved Adhesion and Proliferation of Human Endothelial Cells on Polyethylene Precoated with Monoclonal Antibodies Directed Against Cell Membrane Antigens and Extracellular Matrix Proteins", F.K. Schattauer Verlagsgesellschaft mbH (Stuttgart) 66(6): 715-724 (1991) ("Dekker") in combination with US 5,310,669 to Richmond et al. ("Richmond") and, in some instances, with yet another reference.

6. For the reasons set forth herein, I respectfully yet strongly disagree with the Examiner. At the time the claimed invention was made, a person of ordinary skill in the art would not have been motivated to combine Dekker and Richmond with a reasonable expectation of successfully arriving at the claimed invention.

7. Dekker tried to solve a problem using *ex vivo* "seeded vascular grafts," which is not the subject matter of the claimed invention. Richmond discloses fullerene-coated surfaces such as those of Petri dishes, for use, for example, in culturing homogeneous cell populations *in vitro*. However, there is no suggestion in Richmond to coat a medical device for implantation which is designed to capture progenitor and endothelial cells *in vivo* to form functional endothelium *in situ*.

8. Even the Examiner has acknowledged that the respective disclosures of Dekker and Richmond are limited to *in vitro* studies under static conditions. However, at page 12 of the final Office Action, the Examiner states that combination of prior art, including Dekker and Richmond, would produce a structure that "allows for *in vivo* attachment of cells".

9. At the time the claimed invention was made, a person of ordinary skill in the art would have reasonably expected that shear forces, i.e., *in vivo* under conditions of flow, would quickly wash off any surface-adsorbed antibodies as disclosed by Dekker and Richmond. Therefore, contrary to the Examiner's position, *in vivo* capture of circulating endothelial cells and proliferation of captured cells to bring about an inhibition of restenosis would not and could not have reasonably been expected in view of the prior art. Therefore, it is my opinion that a person of ordinary skill in the art would not have been motivated to combine Dekker and Richmond with a reasonable expectation of successfully producing the claimed medical device for implantation into a patient, wherein the medical device has a coating comprising a matrix layer and antibodies with specificity to bind cell surface antigens of endothelial cells, or progenitors thereof, *in vivo*.

10. Our experiments have surprisingly shown that a single type of antibody which binds a specific cell membrane antigen can be utilized to capture cells *in vitro* or *in vivo*, and support proliferation of the immobilized cells. This is evident by *in vitro* and *in vivo* experiments that my laboratory have performed with different substrates of materials, typically used in medical devices, which were tested using antibodies and matrices of the claimed invention on the following configurations: (a) uncoated material substrates of medical devices; (b) material substrates of medical devices coated with a matrix; and (c) medical devices coated with a matrix and antibody.

11. It took Applicants numerous attempts, and entailed many hours of difficult experimentation, to identify the types of biocompatible matrices as well as the types of antibodies suitable for coating medical devices to promote reendothelialization of an implant at the site of injury and thus prevent restenosis. As stated in the specification, thickening of the blood vessels wall occurs after implantation of a stent due to smooth muscle cell proliferation (See e.g., page 11, lines 7-15).

12. Unexpectedly, our experiments have shown that the claimed medical devices inhibit restenosis. For example, experiments were conducted in which coated and uncoated stents were implanted into the coronary arteries in the hearts of male Yorkshire swine. The implants were left in the swine for a period of 28 days, at which time the arterial segments containing the implants were removed and processed for studies. Histological photomicrographs of cross-sections through coronary arteries of explants from these studies are shown in Figures 1A-1F (attached as Exhibit 2). Figures 1A, 1B and 1C are low magnification showing the circumference of the arteries, whereas Figures 1D, 1E and 1F, respectively, are a higher magnification from experiments of stents (medical devices):

STENT SURFACE CONFIGURATIONS	**CROSS-SECTION THROUGH CORONARY ARTERIES OF EXPLANTS**	
no coating (bare 316L stainless steel stent)	1A	1D
a 316L stainless steel stent coated with a synthetic matrix of the invention, in this case a (carboxymethyl dextran ("Dextran") matrix	1B	1E
a 316L stainless steel stent coated with a Dextran matrix and anti-CD34 antibody	1C	1F

As can be seen from the photomicrographs (Exhibit 2), the stents coated with Dextran and anti-CD34 antibody (Figures 1C and 1F) shows a remarkably thinner blood vessel wall and a larger luminal diameter than the uncoated or Dextran-coated devices (Figures 1A, 1B, 1D and 1E). Attached as Exhibits 3 and 4 are representative scanning electron micrographs of struts from stent explants which had been coated with Dextran, or coated with Dextran and anti-CD34 antibody prior to implantation into a swine artery. As can be seen in the micrographs taken at 1 hour after implantation (Exhibit 3), the Dextran/antibody-coated stent was rapidly covered with progenitor endothelial cells when compared to the stent coated with Dextran alone, and by 48 hours (Exhibit 4), it shows a confluent layer of endothelium when compared to Dextran alone.

13. Similarly, we performed *in vitro* experiments using stainless steel discs coated with a fullerene matrix with or without a layer of anti-CD34 antibodies attached to the fullerene matrix. Progenitor cells isolated from blood donated by healthy human volunteers were isolated with anti-CD34 antibody attached to magnetic beads from the samples, and incubated with the fullerene-coated and fullerene-plus-antibody-coated samples for 7 days. The samples were fixed in 2% paraformaldehyde, washed and stained with a nuclear stain, Propidium iodide, and counter-stained with anti-vascular endothelial growth factor receptor-2 (anti-VEGFR-2, specific to endothelial cells) secondary antibody conjugated with fluorescein isothiocyanate (FITC) and visualized under a confocal microscope. Photomicrographs of each sample were taken. Figures 2A-2D (Exhibit 5) provide representative data from the experiments. As shown in Figures 2A and 2B, the fullerene-coated stainless steel discs did not show progenitor cell attachment to the discs, as no significant VEGFR-2/FITC fluorescence was present on this disc. However, the fullerene-antibody-coated disc in Figures 2C and 2D show numerous adherent cells on the disc as demonstrated by the fluorescence emanating from the cells' surfaces.

14. Moreover, we conducted similar studies using expanded polytetrafluoroethylene (ePTFE) as a substrate coated with biocompatible matrices, including Dextran and gelatin with or without anti-CD34 antibodies. The graft models were incubated with human progenitor cells in an *in vitro* environment and samples were taken at various times post-incubation. Exhibit 6 is a graph showing the number of cells that adhered to the surface of the graft model at the various times. The graph shows that the number of cells attached to the surface of the samples was significantly greater for those samples coated with a matrix and the antibody. Exhibit 7 shows a bar graph of the results in which it is evident that the claimed medical device coated, with a layer of matrix and an anti-CD34 antibody, shows a 5-fold increase in the amount of cells attached on the surface as compared to bare ePTFE.

15. With specific regard to Dekker, a polyethylene substrate is coated with two different types of adsorbed antibodies (Fab fragment), i.e., one antibody against an endothelial cell surface antigen and a second antibody directed against

extracellular matrix protein. Dekker reports that cell proliferation was completely absent and even detachment of the endothelial cells occurs on the polyethylene substrate coated with adsorbed antibodies. Dekker further reports that two types of antibodies are required to obtain endothelial cell proliferation *in vitro* (see page 722; column 1, lines 52-56; column 2, last paragraph; Figs. 5 and 6). Contrary to Dekker, our data show that a single type of antibody which binds a specific cell membrane antigen can be utilized to capture cells *in vitro or in vivo*, and support proliferation of the immobilized cells in both instances. This is evident by *in vitro* and *in vivo* experiments. Additionally, Dekker concluded that the use of whole antibody would not be feasible, since the "[p]latelet activation by adsorbed intact antibody is probably mediated by the Fc parts of the IgG molecules." This is contrary to our findings, as all of the *in vivo* experiments presented herewith were performed using intact antibodies on the coating. As such Dekker can be said to teach away from the claimed invention.

16. With specific regard to Richmond, the disclosure is limited to cell culture substrates for *in vitro* biological applications, e.g., growing cells in Petri dishes. Richmond is silent with respect to any actual medical application, and there is nothing in Richmond to suggest that cells growing on the static culture substrates disclosed by Richmond could withstand shear forces *in vivo* under conditions of flow. Therefore, a person of ordinary skill in the art would reasonably expect that any surface-adsorbed antibodies disclosed by Richmond would be quickly washed off by such *in vivo* shear forces. Accordingly, at the time the claimed invention was made, Richmond did not and could not offer a reasonable expectation of successfully using a fullerene-coated substrate to promote the attachment of endothelial cells *in vivo* in the first place, which attachment would result in proliferation of the cells, and formation of functional endothelium *in vivo*.

17. Furthermore, Richmond does not disclose a fullerene matrix with specific antibodies attached thereto for binding endothelial cells. Our own studies presented above under paragraphs 12-14, particularly those in paragraph 13, show that medical devices having different synthetic or naturally occurring matrices in the coating, for example, Dextran, fullerene and gelatin, in combination with the antibody of the claimed invention resulted in specific binding to an endothelial cell surface antigen, proliferation of the bound cells and inhibition of restenosis. These results were unexpected and not predictable from Richmond, whether taken alone or in combination with Dekker.

18. Finally, our data described in paragraph 13 above show that a single antibody specific to an endothelial cell surface antigen in combination with a fullerene matrix in the coating of the medical device promotes increased binding and proliferation of endothelial cells, and is contrary to the data in Dekker et al. and Richmond et al. which, respectively, assert that two antibody types are needed for *in vitro* cell growth (Dekker at Abstract) and that growth factors are required in the incubation medium to promote *in vitro* cell growth (Richmond et al. col. 3 lines 48-64).

19. In my experience, it is not possible to predict with reasonable certainty the properties that a medical device will exhibit *in vivo* when compared to *in vitro* since the conditions vary tremendously. The fact that the present coating on the medical device yielded the reported results was unexpected and could not have been reasonably predicted.

I further declare that all statements made herein of my own knowledge are true and that all statements made on information and belief are believed to be true; and further that these statements were made with the knowledge that willful false statements, and the like, so made are punishable by fine or imprisonment, or both under Section 1001 of Title 18 of the United States Code, and that such willful false statements may jeopardize the validity of any patent issued thereon.

Signed in full: _____

Michael John Bradley Kutryk

Dated: Oct 17-05.

CURRICULUM VITAE

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A. EDUCATION

1995-1999	Interventional Cardiology Fellow – Post-Doctoral Fellow Clinician/Scientist of the Medical Research Council of Canada University Hospital Rotterdam – Dijkzigt Erasmus University Rotterdam, The Netherlands
1995	Subspecialty Training in Cardiology Royal Victoria Hospital, McGill University Montreal, Canada
1994	Speciality Certification in Internal Medicine Royal Victoria Hospital, McGill University Montreal, Canada
1991	Ph.D. (Cardiovascular Physiology) University of Manitoba Winnipeg, Canada
1990	M.D. University of Manitoba Winnipeg, Canada
1984	Masters of Science (Cardiovascular Physiology) University of Manitoba Winnipeg, Canada

1981	Bachelor of Science (Joint Chemistry and Microbiology) First Class Honors University of Manitoba Winnipeg, Canada

B. AWARDS AND SCHOLARSHIPS

2004	Young Investigator Award - 2004 Canadian Cardiovascular Society Ottawa, Canada
1995-1999	Medical Research Council of Canada Clinician-Scientist Award University Hospital Rotterdam – Dijkzigt Erasmus University Rotterdam, The Netherlands
1995	Paul P. David de Merck Frosst Bourse de Perfectionnement Association des Cardiologues du Quebec Montreal, Canada
1995	Dr. Benjamin Levitan Award Royal Victoria Hospital Montreal, Canada
1991	E.L. Drewry Memorial Award for Research Excellence University of Manitoba Winnipeg, Canada
1991	St. Boniface Hospital Research Foundation Inc. Award for Research Excellence in Cardiovascular Biology Winnipeg, Canada
1988	Excellence in Research Award University of Manitoba Faculty of Medicine Student Research Awards Day Poster Competition Winnipeg, Canada
1987-90	Canadian Heart Foundation Medical Scientist Scholarship Winnipeg, Canada

1985-86	Canadian Heart Foundation Studentship Winnipeg, Canada
1984-85	Manitoba Health Research Council Studentship Winnipeg, Canada
1982-84	Canadian Heart Foundation Studentship Winnipeg, Canada
1980	Proctor and Gamble Award University of Manitoba Winnipeg, Canada
1980-81	Joseph Hogg Scholarship in Chemistry University of Manitoba Winnipeg, Canada

C. RESEARCH GRANTS

2003-present	Heart and Stroke Foundation of Ontario Research Operating Grant Toronto, Ontario

D. RESIDENCIES, FELLOWSHIPS

1990-1993	Resident, Internal Medicine Royal Victoria Hospital Montreal, Canada
1993-1994	Resident, Cardiology Royal Victoria Hospital Montreal, Canada
1994-1995	Chief Resident, Cardiology Royal Victoria Hospital Montreal, Canada
1995-1999	Interventional Cardiology Fellow Post-Doctoral Fellow Molecular Biology University Hospital Rotterdam -Dijkzigt Erasmus University Rotterdam, The Netherlands

1999-2000 Research Fellow, Cardiology
St. Michael's Hospital
Toronto, Canada

2000-2001 Cardiology Resident
University Health Network
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E. TEACHING APPOINTMENTS

1983-1986 Lecturer; Cardiovascular Physiology
School of Medical Rehabilitation
University of Manitoba
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F. PROFESSIONAL AFFILIATIONS

Fellow of the Royal College of Physicians of Canada

International Society for Heart Research; Member

Associate Scientist, R. Samuel McLaughlin Centre for Molecular Medicine

G. EDITORIAL BOARDS

1. Cardiovascular Radiation Medicine, Elsevier Science Inc. New York, NY.

H. PUBLICATIONS

Full Length Papers:

1. Jamieson JC, Kutryk MJB. Studies on the effect of phenylbutazone and salicylate on the rates of synthesis of albumin and acute-phase α_1-acid glycoprotein by rat liver slices. Biochem Med 1981;23:292-301.

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9. Kutryk MJB, Pierce GN, Dhalla NS. Alterations in heart and serum lysosomal activities in streptozotocin-induced diabetes. Basic Res Cardiol 1987;82:271-278.

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18. Kutryk MJB, van Dortmont LMC, van Meegen JR, de Crom RPG, Wulf PS, Visser WJ, Hamming J, van der Giessen WJ, van der Kamp AWM. Endoluminal cell seeding with allogenic endothelial cells. Endothelium 1997;5:357.

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Reviews and Editorials:

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Book Chapters:

1. Kutryk MJB, Serruys PW. Stenting. In: Topol EJ (ed), *Textbook of Cardiovascular Medicine*. Philadelphia: Lippincott-Raven, 1997.

2. Kutryk MJB, Serruys PW. Introduction to coronary artery stenting: background, different

types, newer designs, general principles. In: Pepine CJ (ed), *Diagnostic and Therapeutic Cardiac Catheterization*. Baltimore: Williams and Wilkins, 1998.

3. Kutryk MJB, Serruys PW. Stents: the menu. In Topol EJ (ed), *Textbook of Interventional Cardiology, 3rd edition*. Philadelphia: W.B. Saunders Company, 1998.

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5. Kutryk MJB, Serruys PW. Antirestenosis alternatives for the next millennium. In: Waksman R (ed), *Vascular Brachytherapy, 2nd Edition*. Armonk: Futura Publishing Company, Inc., 1998.

6. Foley DP, Kutryk MJB. Self-expanding stents. In: Ellis S and Holmes D (eds), *Strategic Approaches in Coronary Intervention, 2nd edition*. Philadelphia: Lippincott Williams and Wilkins, 1999.

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9. Kutryk MJB, Serruys PW. Stents: the menu. In: Topol EJ (ed), *Textbook of Interventional Cardiology, 4th edition*. Philadelphia: W.B. Saunders Company, 2002.

10. Kutryk MJB, Stewart DJS. Pharmacologic revascularization. In: Théroux P (ed), *Acute coronary syndromes. A companion to Braunwald's Heart Disease*. Philadelphia: Elsevier Science, 2003.

11. Kutryk MJB. Coronary rupture and tamponade. In: Rothman M (ed), *Case studies in interventional cardiology*. Trowbridge: Cromwell Press, 2004.

12. Kutryk MJB, Serruys PW. Local delivery of antisense oligomers to c-myc for the prevention of restenosis. In: Edoardo Camenzind and Ivan De Scheerder (eds), *Local Drug Delivery for Coronary Artery Disease Established and Emerging Applications*. London: Taylor & Francis Group, 2005.

Books

1. Kutryk MJB, Serruys PW. *Current State of Coronary Stenting*. Rotterdam: Barejesteh van Wallijk van Doorn and Co's Uitgeversmaatshappij, 1997.

2. Serruys PW, Kutryk MJB (eds). *Handbook of Coronary Stents, 2nd edition*. London: Martin Dunitz Ltd., 1998.

3. Kutryk MJB, Serruys PW. *Coronary Stenting: Current Perspectives. A Companion to the Handbook of Coronary Stents.* London: Martin Dunitz Ltd., 1999.

4. Serruys PW, Kutryk MJB (eds). *Handbook of Coronary Stents, 3rd edition*. London: Martin Dunitz Ltd., 2000.

5. Serruys PW, Leon MB, Colombo A, Kutryk MJB (eds). *Coronary Lesions: A Pragmatic Approach*. London: Martin Dunitz Ltd., 2001.

I. INVITED LECTURES

1. Current state of drug delivery. Fifth Meeting of Advanced Interventional Cardiology, Cortina d'Ampezzo, Italy. February 12-15, 1997.

2. Oligonucleotides against c-myc. Third Drug Delivery Meeting, Amsterdam, The Netherlands. February 20-22, 1997.

3. Different types of stents: features and benefits. International Workshop "Coronary Stenting and Remodeling". Positano, Italy. May 2-3, 1997.

4. Local delivery of antisense oligonucleotide LR-3280 against c-myc for the prevention of in-stent restenosis: the Italics trial. IBC's Fourth Annual International Conference on Restenosis. Cambridge MA, USA. October 16-17, 1997.

5. ITALICS (Wallstent and antisense against c-myc); The big parade of stents. Fourth Thoraxcenter Course on Coronary Stenting. Rotterdam, The Netherlands. December 9-13 1997.

6. Trial of beta radiation for restenosis; Eurovegas study (Angiojet); Comparison of stents; BENESTENT: final analysis with cost effectiveness studies; Randomized study of laser wire vs. conventional wires for chronic total occlusions; ITALICS trial - local delivery of placebo vs. antisense to SMC DNA after Wallstent implantation. Thirteenth Annual International Symposium: Interventional Cardiology. Snowmass Village, Colorado. March 1-6, 1998.

7. The concept of the helical DNA ladder design of the R stent. Endovascular Therapy Course (ETC). Paris, France. May 5-8, 1998.

8. The ITALICS study. Hotline Session, European Congress of Cardiology. Vienna, Austria. August 22-26, 1998.

9. The Thoraxcenter "Big Stent Parade": emphasizing "new" stent designs; A cavalcade of the newest stent designs: The Jomed stent family. Faculty, Transcatheter Therapeutics (TCT). Washington DC. October 7-11, 1998.

10. Novelties in stent design. Fifth Heart Center Course on Coronary Stenting. Rotterdam, The Netherlands. December 8-12, 1998.

11. Transmyocardial percutaneous revascularization and angiogenesis in patients unamenable to conventional revascularization, the pharmalogical and genetic approach; Stents for bifurcation and bifurcated stents. Sixth Meeting of Advanced Interventional Cardiology, Cortina d'Ampezzo, Italy. February 17-20, 1999.

12. Dedicated bifurcation stents: a technical overview; Non-FDA approved stents – a view to the future. Faculty, Transcatheter Therapeutics (TCT), Washington DC. September 22-26, 1999.

13. Lesion-specific stenting: the right stent, the right approach. Faculty, Transcatheter Therapeutics (TCT), Washington DC. October 19-22, 2000.

14. Myocardial angiogenesis: overview of the clinical trials. Canadian Cardiovascular Congress, Vancouver, BC. October 29-November 1, 2000.

15. In vivo progenitor cell capture for the accelerated endothelialization of endovascular devices. The Paris Course on Revascularization (PCR), Paris, France. May 21-24, 2002.

16. Myocardial angiogenesis. University of Florida Health Science Center/Jacksonville, May 29, 2002.

17. Recruitment of progenitor endothelial cells. Faculty, Transcatheter Therapeutics (TCT), Washington DC. September 24-28, 2002.

18. The future beyond drug-eluting stents; Proffered paper: Immobilized anti-EPC antibodies and the reconstitution of endothelial cells. Faculty, International Symposium on Endovascular Therapy (ISET), Miami FL. January 19-23, 2003.

19. Immobilized anti-EPC antibodies for the promotion of endothelialization. Faculty, Local Drug Delivery Meeting and Cardiovascular Course on Radiation and Molecular Strategies (LDDR), Geneva Switzerland. January 23-25, 2003.

20. The relative effects of angiopoietin-1 vs. VEGF on collateral flow in a porcine model of chronic ischemia. Faculty, Cardiovascular Radiation Therapy (CRT), Washington DC. January 26-29, 2003.

21. Beyond drug eluting stents: accelerated endothelial coverage; Capturing progenitor endothelial cells: the ultimate stent coating and a novel approach to prevent restenosis. Faculty, The Paris Course on Revascularization (EuroPCR), Paris, France. May 20-23, 2003.

22. How important is stent design in the era of drug eluting stents? Open cells vs closed cells; New possibilities for preventing restenosis I: progenitor cells. Faculty, IX Congress of SOLACI, São Paulo, Brazil. July 23-25, 2003.

23. Endothelial progenitor cell recruitment to accelerate healing and reduce restenosis; Endothelial regeneration is the key! A different approach to restenosis – the pro-healing concept; The Orbus Genous Stent Program: Can rapid endothelization of a stent with progenitor endothelial cells reduce restenosis? Faculty, Transcatheter Therapeutics (TCT), Washington, DC. September 15-19, 2003.

24. EPC capture coating: How far is the future? Faculty, GCI-Global Cardiovascular Interventions, Frankfurt/Main, Germany, November 28-29, 2003.

25. The future beyond drug-eluting stents: Progenitor endothelial cell capture. Faculty, International Symposium on Endovascular Therapy (ISET), Miami FL. January 25-29, 2004.

26. Vascular tolerance to: biological polymer with molecular binding technology. Faculty, Local Drug Delivery Meeting and Cardiovascular Course on Radiation and Molecular Strategies (LDDR), Geneva Switzerland. January 29-31, 2004.

27. The Genous endothelial cell capture approach: Concept, experimental results, and the HEALING I and II clinical trials. Enter The Drug-Eluting Stent Revolution III: Clinical Impact and Evolving Perspectives. American College of Cardiology, New Orleans, LA. March 6, 2004.

28. Endothelial progenitor cell capture on stent platforms: A novel approach to thrombo-resistance and restenosis prevention. Faculty, Nineteenth Annual International Symposium: Interventional Cardiology. Snowmass Village, Colorado. March 22-26, 2004.

29. Judging a stent by its cover: What does the era of coated stents mean for your patient? Faculty, St. Michael's Hospital Cardiology Day. Toronto, Ontario. April 03, 2004

30. A pro-healing approach to stent thrombo-resistance and restenosis prevention: Endothelial progenitor cell capture. Faculty, Angioplasty Summit 2004. Seoul, Korea. April 29-May 1, 2004.

31. Progenitor endothelial cells on a stent: The Orbus Program. Faculty, Cardiovascular Radiation Therapy (CRT), Washington DC. May 5-7, 2004.

32. Accelerated endothelialization by endothelial progenitor cell capture to promote vascular healing: The HEALING clinical trials. Faculty, The Paris Course on Revascularization (EuroPCR), Paris, France. May 25-28, 2004.

33. Stem Cells, EPC and New Pro-Healing Strategies in PCI. Faculty. 13th Interventional Cardiology Live Course, Montreal, Quebec. June 9-11, 2004.

34. A pro-healing approach to thrombo-resistance and restenosis prevention: Endothelial progenitor cell capture; Endothelial progenitor capture for the passivation of endoluminal devices – promotion of vascular healing. Faculty, Tokyo Percutaneous Coronary Intervention Conference (TOPIC), Tokyo, Japan. July 22-24, 2004.

35. Capturing circulation endothelial progenitor cells: A novel approach to prevent thrombosis and minimize restenosis; Beyond DES. Endothelial progenitor cell capture. The GENOUS stent. Faculty, Singapore International Cardiovascular Therapeutics (SICT). Singapore, Republic of Singapore. August 5-7, 2004.

36. How to heal blood vessels. Faculty. International New Cardiovascular Technology Congress (INCTC). Quebec City, Quebec. September 10,11, 2004.

37. Endothelial progenitor capture for the passivation of endoluminal devices - promotion of vascular healing. University Rounds. Toronto, Ontario. October 7, 2004.

38. The future beyond drug-eluting stents: Progenitor endothelial cell capture. Faculty. Singapore Live 2005 – 14th Annual Live Interventions in Vascular Endotherapy. Singapore, Republic of Singapore. January 24-26, 2005.

39. Endothelial progenitor cell capture for the prevention of stent thrombosis and restenosis. Faculty. 11th Local Drug Delivery Meeting and Cardiovascular Course on Radiation and Molecular Strategies (LDDR), Geneva Switzerland. February 3-5, 2005.

40. Genous endothelial progenitor cell capture. Faculty. Enter The Drug-Eluting Stent Revolution IV: A Critical Appraisal. American College of Cardiology, Orlando, FL. March 5, 2005.

41. Endothelial progenitor cell coating. Faculty. Interventional Cardiology 2005: Twentieth Annual International Symposium: Interventional Cardiology. Snowmass Village, Colorado. March 22-26, 2005.

42. Progenitor Cells Healing I and II. Drug Eluting Stents In-Depth. Faculty. Cardiovascular Revascularization Therapies (CRT), Washington, DC. March 28-31, 2005.

43. Stem Cell Therapy: The PHACET Trial. Faculty. Cardiology for the Practitioner: Critical Pathways. Toronto, Ontario. April 16, 2005.

44. Molecular approaches to prevent restenosis using novel stent strategies. National Research Forum for Young Investigators in Circulatory and Respiratory Health. Winnipeg, Manitoba. April 26-May 1, 2005.

J. PATENTS

1. Kutryk MJB, Cottone RJ, Rowland SM. Coating that promotes endothelial cell adherence. IPN: WO 01/68158